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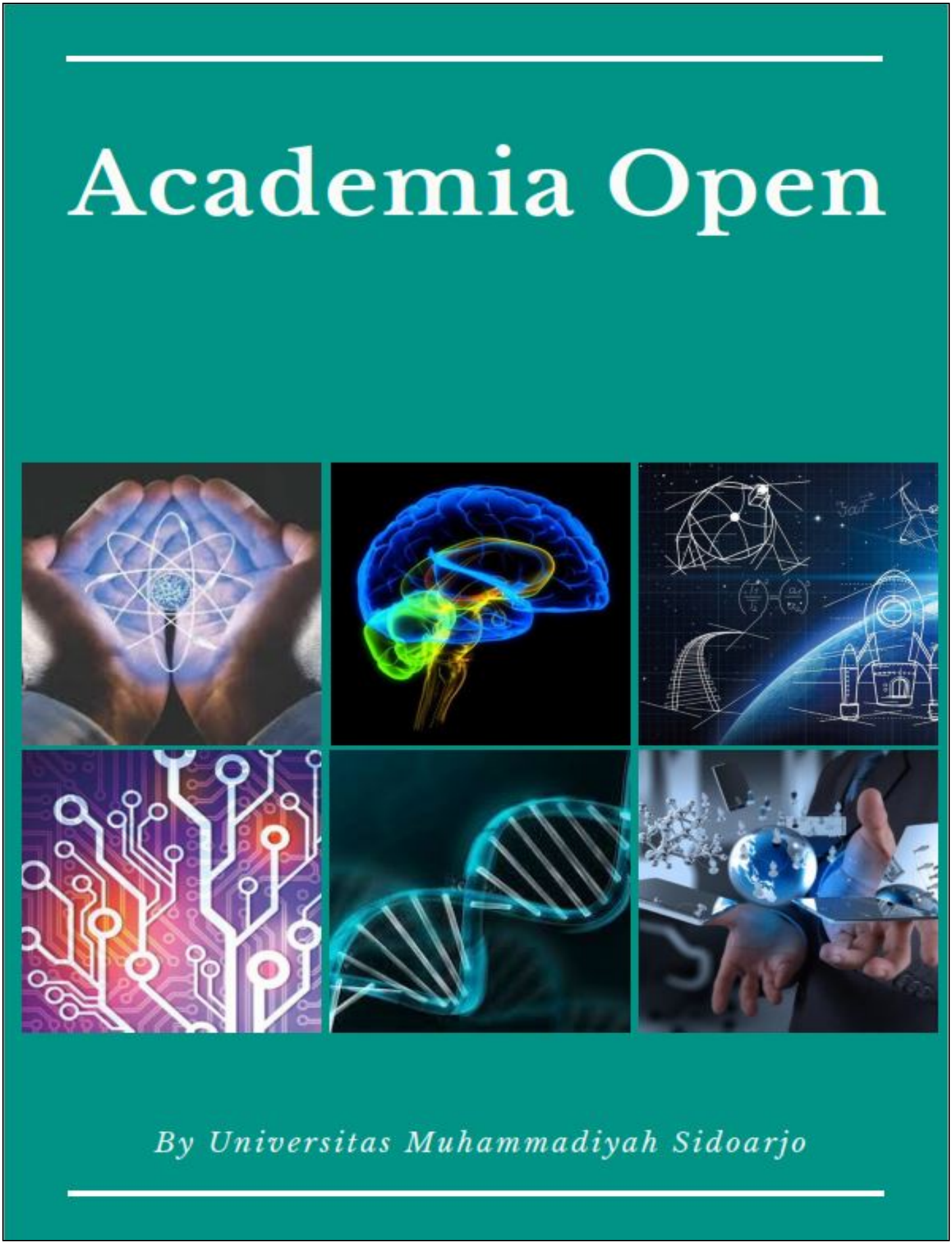

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Chemerin, CK, and LDH as Potential Biomarkers for Early Myocardial Infarction Detection

Chemerin, CK, dan LDH sebagai Biomarker Potensial untuk Deteksi Dini Infark Miokard

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Abstract

General Background: Myocardial infarction (MI) remains a leading cause of mortality worldwide, with early diagnosis being crucial for effective intervention. **Specific Background:** Biomarkers such as Chemerin, Creatine Kinase (CK), and Lactate Dehydrogenase (LDH) have been implicated in inflammatory responses and vascular dysfunction associated with MI. **Knowledge Gap:** Despite existing studies on these biomarkers, their combined diagnostic potential and correlation with cardiovascular risk factors remain underexplored. **Aims:** This study investigates the relationship between Chemerin, CK, and LDH levels in MI patients, alongside other risk factors such as body mass index (BMI) and smoking, to assess their role as potential early diagnostic indicators. **Results:** A total of 70 samples (50 MI patients, 20 controls) were analyzed at Azadi Educational Hospital, Kirkuk, from November 2023 to March 2024. The study found significantly elevated levels of Chemerin, CK, and LDH ($P < 0.05$) in MI patients compared to controls, indicating their involvement in inflammation and myocardial damage. **Novelty:** This study provides evidence that these biomarkers, particularly Chemerin, may serve as predictive indicators for MI risk, offering an alternative approach for early detection. **Implications:** The findings highlight the potential of biomarker-based screening in clinical settings and emphasize the importance of lifestyle modifications to mitigate MI risks and complications.

Highlights:

1. Biomarkers play a crucial role in myocardial infarction diagnosis.
2. Chemerin, CK, and LDH significantly elevated in MI patients ($P < 0.05$).
3. Potential biomarkers for early MI detection and risk assessment.

Keywords: Chemerin; Creatine Kinase; lactate dehydrogenase; Myocardial Infarction; Smoking.

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Introduction

Myocardial infarction, commonly referred to as a heart attack, is the leading cause of mortality worldwide. This critical medical condition arises when blood supply to a section of the heart muscle is obstructed due to a blockage in a coronary artery. The obstruction typically results from a blood clot forming over fatty deposits (plaques) that build up along arterial walls, contributing to atherosclerosis]1[.

When a part of the heart is deprived of oxygen and nutrients due to reduced blood flow, the heart cells in that area begin to die within a period ranging from minutes to hours. The longer the blockage persists without treatment, the greater the area of damage to the heart muscle, which in turn will lead to a weakening of its function or even complete cessation of its work in severe cases, as illustrated in Figure.1 [2[.

The danger of a myocardial infarction lies in the physiological and pathological events that occur in the body after the infarction. These can affect the contraction process of the heart muscle, leading to a decrease in cardiac output and stroke volume. If the infarction persists, it will result in ventricular hypertrophy, which in turn contributes to a reduction in stroke volume, negatively impacting the perfusion of vital organs [3[.

Several factors contribute to the increased risk of heart disease and stroke, with unhealthy lifestyle choices playing a significant role in their development. Poor dietary habits, physical inactivity, smoking, and excessive alcohol intake are key contributors. Additionally, air pollution is a major environmental factor that negatively impacts cardiovascular health..[4]

Figure 1 is here

These factors can lead to health issues such as high blood pressure, increased blood sugar and fat levels, as well as weight gain and obesity, which are known as "intermediate risk factors." These health indicators can be identified in primary care centers, where they are considered early signs of the potential for heart attacks, heart failure, and other serious complications [5[.

Chemerin is a sugary protein consisting of a series of amino acids and contains in its composition 163 amino acids and is produced in the form of Prochemerin, with his own receptors [6[.

Chemerin is a multi -functional protein and has important roles in the body, as it participates in organizing metabolism and infections, in addition to its role in immune responses, as it works to regulate energy and fat metabolism and has an effect also on insulin sensitivity and Chemerin functions regulating blood vessels and blood pressure and as shown in Figure [2[[7].

Figure 2 is here

Methods

2.1. Experiment design and collection sample

The study included 75 samples taken from patients who were transferred to the hospital in the intensive care unit at Kirkuk Education Hospital in Kirkuk City, where the study included 75 sampels of healthy people and patients who are in the hospital's intensive care unit, and samples were divided into 50 models of patients with myocardial infarction and 25 samples of healthy people as a control group. Ordinary test tubes and were celebrated at a temperature of -20 degrees Celestical until the tests are performed.

2.2 Assessment of Chemerin

The level of the chemerin was estimated in the serum of the blood of patients and healthy group using several kit diagnosis, and the plate (ELISA) device is used in advance in the form of a plate consisting of (96) pits and coated with antibodies and samples are placed in the pits where the antigens of the two codes are linked to the pre -installed antibodies, then all the substances are not associated and then the bodies are added. Located with biotin, which is specific to the pits to the drilling, and after the washing of the washing, the associated enzyme (HRP) associated with the Avidin (Avidin) is added to the wells, and it wants to wash again to remove the detector of the beneficiaries of the avidin-enzyme version that is not associated and then the Stop Solution solution is added to stop the action of the enzyme and change the color with what is It fits with the concentration of the associated perishes, after which absorption is read on the (ELISA) device and a 450 NM length and the fields are used to draw Standard Curve.

2.3 Estimation of Creatine Kinase CK activity

The activity of creatine kinase is measured using a coupled enzymatic reaction involving multiple enzymes,

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including creatine kinase, N-acetylcysteine, AMP, ADP, NADP, AP5A, and G6PD, to produce NADPH. This measurement is conducted at a wavelength of 340 nm, where the absorbance is directly proportional to the activity of creatine kinase present in the sample. In this reaction, phosphocreatine and ADP are converted into creatine and ATP. One unit of creatine kinase is defined as the amount of enzyme that transfers one micromole of phosphate from phosphocreatine to ADP per minute at a pH of 6. Based on this method, creatine kinase activity typically ranges from 30 to 1800 U/L.

2.4 Estimation of lactate dehydrogenase LDH activity

The activity of lactate dehydrogenase (LDH) is measured using a hydrogen carrier cofactor, which facilitates the enzymatic reaction. LDH catalyzes the conversion of lactic acid into pyruvate, which then reacts with 2,4-dinitrophenylhydrazine to form pyruvate dinitrophenylhydrazone. This compound exhibits a red to brown coloration in an alkaline solution, with the intensity of the color being directly proportional to the pyruvate concentration. Absorbance measurements are taken at an optimal wavelength of 450 nm, where the absorbance level correlates with the enzyme's activity and efficiency.

2.5 Estimation of Aspartate Aminotransferase (AST/GOT) activity

The activity of aspartate aminotransferase (AST) is measured based on the principle of transamination, where an amino group is transferred between α-ketoglutaric acid and the amino acid aspartic acid. This reaction results in the formation of glutamic acid and oxaloacetic acid. During the process, oxaloacetic acid undergoes decarboxylation to form pyruvic acid, which subsequently reacts with 2,4-dinitrophenylhydrazine (DNPH) to produce 2,4-dinitrophenylhydrazone. This compound exhibits a reddish-brown coloration, with the intensity of the color being directly proportional to the enzyme activity. The absorbance is measured at a wavelength of 340 nm to assess the enzyme's activity.

2.6 Estimation of Alanine Aminotransferase (ALT/GPT) activity

The activity of alanine aminotransferase (ALT) is determined by an enzymatic reaction in which ALT catalyzes the transfer of an amino group from the amino acid alanine to α-ketoglutaric acid, producing pyruvic acid and glutamic acid. This reaction occurs at a physiological pH of 7.4 and a temperature of 37°C. Subsequently, a phenylhydrazine reagent is added, leading to the formation of a phenylhydrazone complex with pyruvic acid. This complex exhibits a reddish-brown coloration in an alkaline medium. The activity of ALT is assessed by measuring absorbance at a wavelength of 510 nm, where absorbance values correlate with enzyme activity.

Result and Discussion

The results in Table 1 showed a significant increase in the levels of both chemerin, Creatine Kinase (CK), and lactate dehydrogenase (LDH) in the group of patients with myocardial infarction compared to the control group.

Parameters	Control Mean ± SD	Patients Mean ± SD	Probability
S.Chemerin (ng/ml)	130 ± 5.6	192 ± 24	≤0.05
Creatine Kinase CK	95.4 ± 7	341 ± 5	≤0.05
lactate dehydrogenase LDH	107 ± 8	480 ± 25	≤0.05

Table 1. *Levels of chemerin, CK and LDH in the patient group compared to the healthy group.*

It is evident from Figure .3 that there is a correlation between elevated levels of chemerin and the occurrence of myocardial infarction, as a significant increase ($p<0.05$) in chemerin levels was found in the affected group (192 ± 24) compared to the control group (130 ± 5.6). This increase may be attributed to the roles of chemerin in the body, one of which is its function as a chemotactic factor for immune cells such as macrophages to the sites of vascular injury, leading to the exacerbation of the local inflammatory response and consequently increasing the risk of plaque rupture, which can cause myocardial infarction]8[. Also, the role of chemerin in stimulating the production of adhesion molecules such as E-selectin, which promotes the adhesion and migration of white blood cells to the vascular wall, leads to increased damage to the vascular endothelium, creating a suitable environment for the development of atherosclerosis and myocardial infarction]9[. In addition to the role of chemerin in stimulating programmed cell death of cardiac muscle cells, this effect may exacerbate cardiac dysfunction during ischemic episodes, making it an important factor in the pathophysiology of myocardial infarction [10).

Figure 3 is here

Figure 4. shows a significant correlation ($p<0.05$) between elevated CK levels and myocardial infarction. The results indicated a significant increase in CK levels in the affected group (341 ± 5) compared to the control group (95.4 ± 7). This correlation may be attributed to several reasons, the most important of which is its presence in

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tissues that require large amounts of energy, such as the heart, muscles, and brain [11] , and its role in energy production within these cells, as it works by converting creatine and phosphate into phosphocreatine, which is a molecule that stores energy for use in the heart muscle tissues. Therefore, its presence in these tissues, which require large amounts of energy, leads to its leakage during a heart attack. The increase in CK levels begins 3-4 hours after a heart attack and then gradually decreases, making it an important indicator in diagnosing the severity of the injury and the time since the injury occurred]12[.

Figure 4 is here

Through Figure (5) a significant increase ($p<0.05$) in LDH levels is observed in the group of patients with myocardial infarction (480 ± 25) compared to the control group (107 ± 8). This enzyme is present in almost all body cells and plays a crucial role in the process of converting pyruvate to lactate during the anaerobic glycolysis of glucose [13[, this increase may be attributed to various reasons, including damage to heart tissues, as seen in myocardial infarction. This leads to the leakage of this enzyme from the damaged cells into the bloodstream. LDH levels increase within 12 hours of the injury and reach their peak within 24-48 hours [14[. Additionally, this injury causes a cessation of blood flow to a part of the heart muscle, resulting in oxygen deficiency and cardiac cell death, and consequently, the leakage of this enzyme, the increase in this enzyme is directly proportional to the extent of damage to the heart muscle cells .[15]

Figure 5 is here

The results in Table 2 showed a significant increase ($p<0.05$) in GOT enzyme levels in the group of patients with myocardial infarction (34 ± 5) compared to the control group (18.4 ± 4), with a significant increase in GPT enzyme levels in the patient group (60.5 ± 4) compared to the control group (32 ± 5.4) .

Parameters	Control Mean ± SD	Patients Mean ± SD	Probability
GOT	18.4 ± 4	34 ± 5	≤0.05
GPT	32 ± 5.4	60.5 ± 4	≤0.05

Table 2. *Levels of liver enzymes GOT and GPT in the patient group compared to the control group.*

Figure (6) shows elevated levels of GOT and GPT enzymes in patients with myocardial infarction compared to healthy individuals. This elevation may be due to these enzymes being present in the liver at high concentrations, reaching approximately 3000 times more than their levels in the blood [16[. Therefore, during a heart attack, there is a decrease in blood and oxygen flow to the body's organs, including the liver, which may lead to ischemia in the liver [shock liver). As a result, liver cells are damaged, and GOT is released into the blood [17[. The cause of this increase may be due to the accumulation of blood in the blood vessels because of the weakened pumping of the heart, leading to heart congestion, cell damage, and an increase in the secretion of these enzymes in the blood [18], In addition, myocardial infarction leads to a systemic inflammatory response that increases pro-inflammatory cytokines such as TNF-α and IL-6, resulting in oxidative stress on the liver and consequently cell damage, causing these enzymes to leak into the bloodstream [19[.

Figure 6 is here

The results in Table 3 showed a significant increase ($p<0.05$) in lipid profile levels in the group of patients with myocardial infarction compared to the control group, except for HDL-C, where the results showed a significant increase in its levels in the control group (40.5 ± 4) compared to the patient group (6 ± 17.7).

Parameters	Control Mean ± SD	Patients Mean ± SD	Probability
S. T- cholesterol (mg/dl)	165 ± 15	205 ± 30	≤0.05
S.TG (mg/dl)	70 ± 7	175 ± 5	≤0.05
S.HDL-C (mg/dl)	40.5 ± 4	17.7 ± 6	≤0.05
S.LDL-C (mg/dl)	75 ± 14	281 ± 41	≤0.05
S. VLDL -C (mg/dl)	16.7 ± 4	43.4 ± 5	≤0.05

Table 3. *Levels of liver enzymes GOT and GPT in the group of patients with myocardial infarction compared to the control group.*

Figure (7) illustrates a notable increase in cholesterol levels among myocardial infarction patients compared to healthy individuals. Cholesterol is a vital component involved in bile acid and bilirubin formation, as well as the synthesis of steroids and vitamin D. It plays a key role in regulating cell membrane fluidity and stability. Maintaining cholesterol homeostasis is essential for both cellular and systemic functions, as its levels are influenced by a balance between production, absorption, export, and esterification. For storage or secretion within

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lipoproteins, cholesterol is converted into neutral cholesteryl esters. This waxy substance is present in both the bloodstream and the human body, and it is needed for cell building. However, elevated cholesterol levels in the blood increase the risk of heart disease because its increase can lead to the formation of fatty deposits in the blood vessels. Over time, these deposits can narrow the arteries and reduce blood flow. In some cases, these deposits may suddenly rupture, which can cause a heart attack or stroke [21[.

The results showed an increase in triglyceride, LDL, and VLDL levels in the affected individuals, and this increase in fats can be attributed to several reasons, including the body's secretion of inflammatory cytokines during a myocardial infarction, which stimulates the liver to produce more fats than usual [22[.

It was found through the results an increase in the levels of triglycerides, LDL and VLDL in the infections and that the increase in these fats can be due to several reasons, including the body's secretion of inflammatory cytokins during the heart muscle infarction, which stimulates the liver to produce fats more than usual [22[, as well as increasing the activity of the friendly nervous system and increasing the secretion of adrenaline leads to the decomposition of fat stored in the fatty tissue Which leads to the increase in free fatty acids, which the liver converts into triple fat [23[, in addition to the lack of liver irrigation during the heart muscle infarction causes a functional defect in the liver and increases the production of these fats and reduces the body's ability to get rid of it [24[.

HDL is very important to its role in protecting the heart and blood vessels, as it works to remove excess cholesterol from tissues and transfer it to the liver for its secretion [25[, and that its decrease in people with heart muscle infarction may be due to several reasons, including high levels of inflammation and oxidative stress in people, which negatively affects the HDL function and reduces its efficiency in removing excess cholesterol from cells [26[.

Figure 7 is here

The findings presented in Table (4) reveal a significant increase in most indicators among myocardial infarction patients compared to the control group. These results highlight the importance of body mass index as a key factor in evaluating the risk of myocardial infarction. Additionally, individuals with abdominal obesity face a higher likelihood of developing various health conditions, including myocardial infarction, and experience elevated rates of morbidity and mortality [27[, this can be attributed to the fact that obesity and overweight occur due to an abnormal accumulation of fat that is harmful to health. One of the main causes of this is the imbalance in calorie consumption with the expenditure of very few of these calories. Among the key factors leading to weight gain are the excessive consumption of calorie-rich, fatty, and sugary foods, and the high rate of physical inactivity due to inactive lifestyles and changes in modes of transportation. The World Health Organization's report indicates that obesity rates have increased significantly worldwide, nearly tripling since 1975. In 2022, there was an increase in obesity rates among adults and adolescents globally, with adult obesity rates doubling since 1990 and quadrupling among adolescents [28[.

Parameters	Groups (Smoking)	Control Mean ± SD	Patients Mean ± SD	Probability	Probability in patients group ≤
S.Chemerin (ng/ml)	15-25	110 ± 7.9	170 ± 25	≤0.05	0.05
	26-35	155 ± 6.3	210 ± 54	≤0.05	
CK (IU/L)	15-25	89 ± 12.9	310 ± 32	≤0.05	0.05
	26-35	124 ± 6.5	387 ± 25	≤0.05	
LDH (IU/L)	15-25	90 ± 7.9	423 ± 31	≤0.05	0.05
	26-35	132 ± 32	492 ± 42	≤0.05	
GOT (IU/L)	15-25	17 ± 9	33 ± 3.6	≤0.05	0.05
	26-35	19.6 ± 6	41 ± 6.5	≤0.05	
GPT (IU/L)	15-25	31 ± 7.4	68 ± 4.7	≤0.05	0.5
	26-35	34 ± 6	78 ± 8.8	≤0.05	
S. T- cholesterol (mg/dl)	15-25	170 ± 23	230 ± 34	≤0.05	0.05
	26-35	176 ± 27	265 ± 34	≤0.05	
S.TG (mg/dl)	15-25	71 ± 9	153 ± 12	≤0.05	0.05
	26-35	81 ± 12	182 ± 6	≤0.05	
S.HDL-C (mg/dl)	15-25	44.5 ± 6	19.5 ± 4	≤0.05	0.4
	26-35	34 ± 3	20 ± 6	≤0.05	
S.LDL-C (mg/dl)	15-25	70 ± 9	275 ± 34	≤0.05	0.05
	26-35	91 ± 8	318 ± 23	≤0.05	
S.VLDL-C (mg/dl)	15-25	17 ± 3.1	44.9 ± 6	≤0.05	0.4
	26-35	18 ± 3.6	49.7± 7	≤0.05	

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Table 4. *Body mass index in the patient group compared to the control group.*

It was found that an increase in body mass index is closely associated with damage to the heart and blood vessels. Among the mechanisms through which obesity causes hypertension are the hyperactivity of the sympathetic nervous system, the stimulation of the renin-angiotensin-aldosterone system, changes in adipose-derived cytokines, and structural and functional renal changes]29][30[. In addition, individuals suffering from severe obesity exhibit an increase in the volume of adipose tissues, which can cause endocrine dysfunctions and increased insulin resistance in the body. This resistance raises the levels of triglycerides and glucose in the blood and leads to high blood pressure, increasing the likelihood of cardiovascular diseases]31[.

Parameters	Groups (Smoking)	Control Mean ± SD	Patients Mean ± SD	Probability	Probability in patients group ≤
S.Chemerin (ng/ml)	smokers	125 ± 5.1	184 ± 25	≤0.05	0.05
	Non-smokers	140 ± 4.3	205 ± 32	≤0.05	
CK	smokers	85.4 ± 9	312 ± 11	≤0.05	0.05
(IU/L)	Non-smokers	105 ± 9	379 ± 22	≤0.05	
LDH	smokers	98 ± 8	465 ± 22	≤0.05	0.2
(IU/L)	Non-smokers	121 ± 21	498 ± 43	≤0.05	
GOT	smokers	17.4 ± 5	31 ± 2	≤0.05	0.05
(IU/L)	Non-smokers	21 ± 3	38 ± 7	≤0.05	
GPT	smokers	28 ± 4.4	63 ± 3	≤0.05	0.05
(IU/L)	Non-smokers	38 ± 3	82 ± 11	≤0.05	
S. T- cholesterol (mg/dl)	smokers	165 ± 15	205 ± 30	≤0.05	0.05
	Non-smokers	180 ± 32	287 ± 54	≤0.05	
S.TG (mg/dl)	smokers	64 ± 4	168 ± 5	≤0.05	0.4
	Non-smokers	79 ± 11	187 ± 9	≤0.05	
S.HDL-C (mg/dl)	smokers	42.5 ± 3	20.7 ± 2	≤0.05	0.5
	Non-smokers	36 ± 8	19 ± 3	≤0.05	
S.LDL-C (mg/dl)	smokers	71 ± 8	274 ± 34	≤0.05	0.05
	Non-smokers	87 ± 21	301 ± 26	≤0.05	
S.VLDL-C (mg/dl)	smokers	16.1 ± 4.1	39.4 ± 3	≤0.05	0.05
	Non-smokers	19 ± 3	51± 4	≤0.05	

Table 5. *The effect of smoking in the group of patients with myocardial infarction compared to the control group*

It is evident from the results in Table (5) that there is a significant increase in most indicators among the group of smoking patients with myocardial infarction compared to the control group. This indicates that smoking is one of the main risk factors for myocardial infarction, which may be due to several reasons, including the presence of many toxic substances in cigarette smoke such as carbon monoxide, nicotine, and free radicals]32[. In addition to that smoking stimulates the production of fibrinogen and increases the adhesion of platelets, which leads to the occurrence of clots that may cause a blockage in the coronary arteries and thus the occurrence of myocardial infarction]33[, as well as the effect of the carbon oxide found in the cigarettes on the efficiency of hemoglobin on the transmission of oxygen, as the corpus of carbon oxide with hemoglobin is stronger than oxygen that reduces blood ability Transfer of oxygen to the heart muscle, which exacerbates the lack of perfusion and increases the risk of myocardial infarction]34[.

Conclusion

Conclude from this study the existence of a moral correlation between the high levels of Chemerin, Creatine Kinase (CK) and Lactate Dehydrogenase (LDH) and increased risk of heart muscle infarction, as it turns out as important factors in pathological physiology for the heart muscle infarction. And the cause of a dysfunction in the vascular lining, in addition to the high CK and LDH reflects the damage of the heart tissue, as well as the effects caused by other studied factors such as smoking and body mass index, and thus these indicators can be used to assess the risks of heart disease, their development and their timing.

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